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Term	Documents
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USPT,PGPB,JPAB,EPAB,DWPI	l1 same l2 same l3	9	L4
USPT,PGPB,JPAB,EPAB,DWPI	glycosyl$10	16697	L3
USPT,PGPB,JPAB,EPAB,DWPI	mutein or substitut$5	776258	L2
USPT,PGPB,JPAB,EPAB,DWPI	fibroblast adj growth adj factor	4165	L1

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Search Results - Record(s) 1 through 9 of 9 returned.

☐ 1. Document ID: US 6080407 A

L4: Entry 1 of 9 File: USPT Jun 27, 2000

US-PAT-NO: 6080407
DOCUMENT-IDENTIFIER: US 6080407 A

TITLE: Diagnostic assays for MIF

DATE-ISSUED: June 27, 2000

NR

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Bucala; Richard J.	New York	NY	N/A	N/A
Mitchell; Robert A.	New York	NY	N/A	N/A
Bernhagen; Jurgen	New York	NY	N/A	N/A
Calandra; Thierry F.	New York	NY	N/A	N/A
Cerami; Anthony	Shelter Island	NY	N/A	N/A

US-CL-CURRENT: 424/158.1; 424/145.1, 424/198.1, 514/169, 530/387.3, 530/388.23, 530/389.2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw. Desc | Image

☐ 2. Document ID: US 6030615 A

L4: Entry 2 of 9 File: USPT Feb 29, 2000

US-PAT-NO: 6030615
DOCUMENT-IDENTIFIER: US 6030615 A

TITLE: Combination method for treating diseases caused by cytokine-mediated toxicity

DATE-ISSUED: February 29, 2000

NR

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Bucala; Richard J.	New York	NY	N/A	N/A
Mitchell; Robert A.	New York	NY	N/A	N/A
Bernhagen; Jurgen	New York	NY	N/A	N/A
Calandra; Thierry F.	New York	NY	N/A	N/A
Cerami; Anthony	Shelter Island	NY	N/A	N/A

US-CL-CURRENT: 424/145.1; 424/154.1, 424/156.1, 424/158.1, 424/172.1, 424/85.2, 530/351, 530/387.1, 530/388.1, 530/388.23, 530/388.24, 530/388.75, 530/389.2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw. Desc | Image

☐ 3. Document ID: US 5464943 A

L4: Entry 3 of 9 File: USPT Nov 7, 1995

US-PAT-NO: 5464943
DOCUMENT-IDENTIFIER: US 5464943 A

TITLE: DNA encoding glycosylated FGF and production thereof

DATE-ISSUED: November 7, 1995

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Senoo; Masaharu	Toyonaka	N/A	N/A	JPX
Sasada; Reiko	Kyoto	N/A	N/A	JPX
Igarashi; Koichi	Kyoto	N/A	N/A	JPX

div of 5,360,896 of record

US-CL-CURRENT: 536/23.5; 435/252.3, 435/252.33, 435/255.1, 435/320.1, 435/69.1, 435/69.4, 530/399, 536/23.51

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

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☐ 4. Document ID: US 5360896 A

L4: Entry 4 of 9 File: USPT Nov 1, 1994

US-PAT-NO: 5360896
DOCUMENT-IDENTIFIER: US 5360896 A

TITLE: Glycosylated FGF

DATE-ISSUED: November 1, 1994

previously cited

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Senoo; Masaharu	Osaka	N/A	N/A	JPX
Sasada; Reiko	Kyoto	N/A	N/A	JPX
Igarashi; Koichi	Kyoto	N/A	N/A	JPX

US-CL-CURRENT: 530/399; 435/69.1, 435/69.4, 435/69.5, 530/397

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

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☐ 5. Document ID: US 5130418 A

L4: Entry 5 of 9 File: USPT Jul 14, 1992

US-PAT-NO: 5130418
DOCUMENT-IDENTIFIER: US 5130418 A

TITLE: Method to stabilize basic fibroblast growth factor

DATE-ISSUED: July 14, 1992

checked KWIC NR

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Thompson; Stewart A.	Mountain View	CA	N/A	N/A

US-CL-CURRENT: 530/399; 530/350, 530/402

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

☐ 6. Document ID: US 5464943 A

L4: Entry 6 of 9 File: EPAB Nov 7, 1995

PUB-NO: US005464943A
DOCUMENT-IDENTIFIER: US 5464943 A
TITLE: DNA encoding glycosylated FGF and production thereof

PUBN-DATE: November 7, 1995

INVENTOR-INFORMATION:

NAME	COUNTRY
SENOO, MASAHARU	JP
SASADA, REIKO	JP
IGARASHI, KOICHI	JP

div of 5,360,896 of record

INT-CL (IPC): C07H 21/00; C07K 14/50; C12N 15/18; C12N 15/63
EUR-CL (EPC): C07K014/50; C07K014/50

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

☐ 7. Document ID: US 5360896 A

L4: Entry 7 of 9 File: EPAB Nov 1, 1994

PUB-NO: US005360896A
DOCUMENT-IDENTIFIER: US 5360896 A
TITLE: Glycosylated FGF

PUBN-DATE: November 1, 1994

INVENTOR-INFORMATION:

NAME	COUNTRY
SENOO, MASAHARU	JP
SASADA, REIKO	JP
IGARASHI, KOICHI	JP

of record

INT-CL (IPC): C07K 13/00
EUR-CL (EPC): C07K014/50

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

☐ 8. Document ID: US 5360896 A

L4: Entry 8 of 9 File: DWPI Nov 1, 1994

of record

DERWENT-ACC-NO: 1994-349502
DERWENT-WEEK: 199443
COPYRIGHT 2001 DERWENT INFORMATION LTD

TITLE: Muteins of naturally occurring fibroblast growth factor - into which have been introduced at least one glycosylation site.

INVENTOR: IGARASHI, K; SASADA, R ; SENOO, M

PRIORITY-DATA: 1990JP-0108595 (April 26, 1990)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 5360896 A	November 1, 1994	E	031	C07K013/00

INT-CL (IPC): C07K 13/00

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Clip Img | Image

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☐ 9. Document ID: EP 394951 A, CA 2015313 A, JP 03061494 A, US 5464943 A

L4: Entry 9 of 9 File: DWPI Oct 31, 1990

DERWENT-ACC-NO: 1990-328987
DERWENT-WEEK: 199044
COPYRIGHT 2001 DERWENT INFORMATION LTD

TITLE: New fibroblast growth factor mutein(s) containing glycosylation site - used for treatment of thrombosis etc., with greater activity and stability than natural FGF

INVENTOR: IGARASHI, K; SASADA, R ; SENOO, M

PRIORITY-DATA: 1989JP-0108595 (April 26, 1989), 1990JP-0109014 (April 25, 1990)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
EP 394951 A	October 31, 1990	N/A	000	N/A
CA 2015313 A	October 26, 1990	N/A	000	N/A
JP 03061494 A	March 18, 1991	N/A	000	N/A
US 5464943 A	November 7, 1995	N/A	031	C07H021/00

related to 5,360,896 of record

INT-CL (IPC): A61K 37/36; C07H 21/00; C07K 13/00; C07K 14/50; C12N 1/00; C12N 5/10; C12N 15/16; C12N 15/18; C12N 15/63; C12N 21/00; C12P 21/02; C12R 1/91

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

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```
=> s fibroblast(W)growth(W)factor
          60398 FIBROBLAST
          54193 FIBROBLASTS
          81765 FIBROBLAST
                  (FIBROBLAST OR FIBROBLASTS)
         884916 GROWTH
           2769 GROWTHS
         886239 GROWTH
                  (GROWTH OR GROWTHS)
         634753 FACTOR
         521042 FACTORS
         988903 FACTOR
                  (FACTOR OR FACTORS)
L1        12666 FIBROBLAST(W)GROWTH(W)FACTOR

=> s mutein or variant or substitut?

            301 MUTEIN
            291 MUTEINS
            438 MUTEIN
                  (MUTEIN OR MUTEINS)
          39447 VARIANT
          39784 VARIANTS
          68440 VARIANT
                  (VARIANT OR VARIANTS)
         614709 SUBSTITUT?
L2       676825 MUTEIN OR VARIANT OR SUBSTITUT?

=> s glycosylat

=> s glycosylat

=> s glycosyl?

L3        43855 GLYCOSYL?

=> s l1 and l2 and l3

L4            9 L1 AND L2 AND L3

=> d l4 1-9 bib ab
```

L4 ANSWER 1 OF 9 CA COPYRIGHT 2001 ACS
AN 133:115253 CA
TI **Fibroblast growth factor** (FGF) receptor 1-IIIb is a naturally occurring functional receptor for FGFs that is preferentially expressed in the skin and the brain
AU Beer, Hans-Dietmar; Vindevoghel, Laurence; Gait, Mary J.; Revest, Jean-Michel; Duan, D. Roxanne; Mason, Ivor; Dickson, Clive; Werner, Sabine
CS Institute of Cell Biology, Swiss Federal Institute of Technology, Zurich, CH-8093, Switz.
SO J. Biol. Chem. (2000), 275(21), 16091-16097
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB **Fibroblast growth factors** (FGFs) transmit their signals through four transmembrane receptors that are designated FGFR1-4. Alternative splicing in the extracellular region of FGFR1-3 generates receptor **variants** with different ligand binding affinities. Thus two types of transmembrane receptors (IIIb and IIIc isoforms) have been identified for FGFR2 and FGFR3, and the existence of analogous **variants** has been postulated for FGFR1 based on its genomic structure. However, only a single full-length transmembrane FGFR1 **variant** (FGFR1-IIIc) has been identified so far. Here the authors describe the cloning of a full-length cDNA encoding FGFR1-IIIb from a mouse skin wound cDNA library. This receptor isoform was expressed at the highest levels in a subset of sebaceous glands of the skin and in neurons of the hippocampus and the cerebellum. FGFR1-IIIb was expressed in L6 rat skeletal muscle myoblasts and used in crosslinking and receptor binding studies. FGF-1 was found to bind the receptor with high affinity, whereas FGF-2, -10, and -7 bound with significantly lower affinities. Despite their apparently similar but low affinities, FGF-10 but not FGF-7 induced the activation of p44/42 mitogen-activated protein kinase in FGFR1-IIIb-expressing L6 myoblasts and stimulated mitogenesis in these cells, demonstrating that this new receptor **variant** is a functional transmembrane receptor for FGF-10.
RE.CNT 47
RE
(1) Basilico, C; Adv Cancer Res 1992, V59, P115 CA
(2) Beer, H; Oncogene 1997, V15, P2211 CA
(3) Burrus, L; Mol Cell Biol 1992, V12, P5600 CA
(4) Chellaiah, A; J Biol Chem 1994, V269, P11620 CA
(5) Chellaiah, A; J Biol Chem 1999, V274, P34785 CA
ALL CITATIONS AVAILABLE IN THE RE FORMAT

NR

L4 ANSWER 2 OF 9 CA COPYRIGHT 2001 ACS
AN 132:31744 CA
TI Gene probes used for genetic profiling in healthcare screening and planning
IN Roberts, Gareth Wyn
PA Genostic Pharma Ltd., UK
SO PCT Int. Appl., 745 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9964627	A2	19991216	WO 1999-GB1780	19990604

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ,

DE, DK, [redacted]ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, [redacted], KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRAI GB 1998-12099 A 19980606
GB 1998-13291 A 19980620
GB 1998-13611 A 19980624
GB 1998-13835 A 19980627
GB 1998-14110 A 19980701
GB 1998-14580 A 19980707
GB 1998-15438 A 19980716
GB 1998-15574 A 19980718
GB 1998-15576 A 19980718
GB 1998-16085 A 19980724
GB 1998-16086 A 19980724
GB 1998-16921 A 19980805
GB 1998-17097 A 19980807
GB 1998-17200 A 19980808
GB 1998-17632 A 19980814
GB 1998-17943 A 19980819

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response.

In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the no. of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide crit. clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies which comprises of the identification of the core group of genes and their sequence **variants** required to provide a broad base of clin. prognostic information - "genostics". The "Genostic.RTM." profiling of patients and persons will radically enhance the ability of clinicians, healthcare professionals and other parties to plan and manage healthcare provision and the targeting of appropriate healthcare resources to those deemed most in need. The use of this invention could also lead to a host of new applications for such profiling technologies, such as identification of persons with particular work or environment related risk, selection of applicants for employment, training or specific opportunities or for the enhancing of the planning and organization of health services, education services and social services.

L4 ANSWER 3 OF 9 CA COPYRIGHT 2001 ACS
AN 132:31743 CA
TI Gene probes used for genetic profiling in healthcare screening and planning
IN Roberts, Gareth Wyn
PA Genostic Pharma Limited, UK

SO PCT Int. Appl., pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9964626	A2	19991216	WO 1999-GB1779	19990604
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9941586	A1	19991230	AU 1999-41586	19990604
AU 9941587	A1	19991230	AU 1999-41587	19990604
GB 2339200	A1	20000119	GB 1999-12914	19990604
EP 1084273	A1	20010321	EP 1999-925207	19990604
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRAI GB 1998-12098 A 19980606
GB 1998-28289 A 19981223
GB 1998-16086 A 19980724
GB 1998-16921 A 19980805
GB 1998-17097 A 19980807
GB 1998-17200 A 19980808
GB 1998-17632 A 19980814
GB 1998-17943 A 19980819
WO 1999-GB1779 W 19990604

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response.

In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the no. of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide crit. clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies.

L4 ANSWER 4 OF 9 CA COPYRIGHT 2001 ACS
AN 127:325826 CA
TI Heparan sulfate - a polyanion with multiple messages
AU Lindahl, Ulf
CS Dept. of Medical and Physiological Chemistry, University of Uppsala, Uppsala, S-751 23, Swed.
SO Pure Appl. Chem. (1997), 69(9), 1897-1902
CODEN: PACHAS; ISSN: 0033-4545
PB Blackwell
DT Journal; General Review
LA English

AB A review with 29 refs. Proteoglycans are composed of sulfate-**substituted**, neg. charged glycosaminoglycan chains that are covalently linked to proteins. Studies on proteoglycan biosynthesis have been focused on the isolation and mol. cloning of the various enzymes that catalyze this process. Enzymes involved in the biosynthesis of heparin and heparan sulfate include the **glycosyltransferases** responsible for generating the initial (GlcA-GlcNAc)n chains, the GlcNAc N-deacetylase/N-sulfotransferase that introduces N-sulfate groups, the D-GlcA C5-epimerase that generates L-IdoA units, and O-sulfotransferases that sulfate hydroxyl groups in various positions. Restricted polymer modification will lead to the generation of complex saccharide sequences of varied structure. Attempts have been made to define the minimal saccharide sequences required for binding of various proteins of biol. interest, including growth factors of the **fibroblast growth factor** family. It is proposed that many "heparin-binding proteins", with affinity for the predominant structure in the highly sulfated heparin mol., may bind to distinct, less modified, regions of heparan sulfate chains. These studies are expected to promote our understanding of the regulatory mechanisms behind polysaccharide biosynthesis, and of the physiol. roles of proteoglycans. Further, they may provide the basis for the generation of novel drugs.

NR

L4 ANSWER 5 OF 9 CA COPYRIGHT 2001 ACS
AN 126:29532 CA
TI A proteoglycan that activates **fibroblast growth factors** during early neuronal development is a perlecan **variant**
AU Joseph, Sharon J.; Ford, Miriam D.; Barth, Christian; Portbury, Stuart; Bartlett, Perry F.; Nurcombe, Victor; Greferath, Ursula
CS Dep. Anatomy Cell Biology, Univ. Melbourne, Parkville, 3052, Australia
SO Development (Cambridge, U. K.) (1996), 122(11), 3443-3452
CODEN: DEVPED; ISSN: 0950-1991
PB Company of Biologists
DT Journal
LA English
AB Cells in the early embryonic vertebrate nervous system are dependent on members of the **fibroblast growth factor** (FGF) family for their proliferation and subsequent differentiation. These growth factors will only bind to their specific high affinity cell surface receptors after formation of a ternary complex with the glycosaminoglycan heparan sulfate. Such specific heparan sulfates are secreted as proteoglycans from neural precursor cells and localize to their surfaces. One such proteoglycan, HSPG-PRM (Perlecan-related mol.), was isolated through its ability to potentiate neural cell responses to either FGF-1, or FGF-2. In this study, we have verified the relative mol. mass of the core protein of PRM as 45,000 and obtained partial amino acid sequence from it. The sequences bore significant homol. to native perlecan. A probe generated by reverse transcriptase polymerase chain reaction using oligonucleotides designed from the protein sequence used on northern blots of RNA from a neuroepithelial cell line detected perlecan at 12.6 kilobases, as well as novel transcripts at 6.5 and 3.5 kilobases. The latter species appears by virtue of its size and abundance to be the novel PRM transcript. PRM appears to be encoded by the same gene as perlecan, as genomic Southern blotting only detected a single gene. Polyclonal antibodies raised against the PRM mol. detected a single proteoglycan species at 290 .times. 103 with a core protein of 45 .times. 103. Polyclonal anti-perlecan antibodies cross-reacted with PRM confirming their relatedness, although immunohistochem. studies revealed a differential staining pattern for PRM as compared to perlecan within the developing nervous system. The PRM mol. was shown to be localized to

NR

several different tissues of the developing embryo indicating that it plays a broad r[redacted] We conclude that PRM is a **var[redacted]nt** of perlecan that is differentially **glycosylated** in a manner that confers highly specific functions at crit. stages of neural development and tissue growth.

L4 ANSWER 6 OF 9 CA COPYRIGHT 2001 ACS
AN 125:318046 CA
TI Identification and characterization of a novel, intracellular isoform of **fibroblast growth factor** receptor-1 (FGFR-1)
AU Maher, Pamela A.
CS Dep. Cell Biol., Scripps Res. Inst., La Jolla, CA, 92037, USA
SO J. Cell. Physiol. (1996), 169(2), 380-390
CODEN: JCLLAX; ISSN: 0021-9541
DT Journal
LA English
AB A novel, low mol. wt., intracellular isoform of FGF receptor-1 (FGFR-1) was identified in embryonic chicken tissues using several antibodies specific for different domains of FGF receptors. This low mol. wt. isoform differs from the previously characterized isoforms of FGFR-1 in that it contains little or no carbohydrate. Furthermore, in contrast to the other isoforms of FGFR-1, this novel isoform is located exclusively intracellularly. However, it is capable of binding 125I-FGF-2 and it possesses intrinsic kinase activity. Pulse-chase expts. indicate that this isoform of FGFR-1 is not simply a precursor to **glycosylated** FGFR-1 since it can be detected long after the appearance of **glycosylated** FGFR-1 in the cells. These results suggest that the novel FGFR-1 isoform plays a role in regulating FGF activity distinct from cell surface, **glycosylated** FGFR-1. The possible roles of this FGFR-1 **variant** in FGF signaling are discussed.

NR

L4 ANSWER 7 OF 9 CA COPYRIGHT 2001 ACS
AN 122:46716 CA
TI Effect of cysteine **substitutions** on the mitogenic activity and stability of recombinant human keratinocyte growth factor
AU Bare, lance A.; Brown, Marlene; Goyal, Shefali; Idler, Denise; Mansson, Per-Erik
CS Ohmeda, PPD, Murray Hill, NJ, 07974, USA
SO Biochem. Biophys. Res. Commun. (1994), 205(1), 872-9
CODEN: BBRCA9; ISSN: 0006-291X
DT Journal
LA English
AB Human Keratinocyte growth factor (hKGF), a member of the FGF family of growth factors, contains five cysteines at amino acid positions 1, 15, 40, 102, and 106. The authors expressed five cysteine mutants of hKGF in which the cysteines were cumulatively replaced with alanine or serine, starting with cysteine-1. Recombinant hKGF has an inherently higher mitogenic activity and stability to heat and acid than reported for **glycosylated** hKGF. Mitogenic activity is increased an addnl. 2.6 fold by **substitution** of cysteine-1 with alanine. Mutants with the conserved cysteine **substituted** at position 40 were more susceptible to heat inactivation than rhKGF, but showed no significant difference in acid inactivation. Cysteine-free rhKGF is mitogenic, demonstrating that neither cysteines nor disulfide bonds are required for mitogenic activity. However, cysteine-free rhKGF does not bind heparin-Sepharose and is unstable to heat and acid compared to rhKGF, suggesting that the cysteines have a role in maintaining KGF's structure. This information will useful in the development of a more stable and more potent wound healing agent from hKGF.

L4 ANSWER 8 OF 9 CA COPYRIGHT 2001 ACS
AN 120:261727 CA
TI An endogenous **glycosylphosphatidylinositol**-specific

phospholipase D releases basic **fibroblast growth factor**-heparan sulfate proteoglycan complexes from human bone marrow cultures

AU Brunner, Georg; Metz, Christine N.; Nguyen, Hiep; Gabrilove, Janice; Patel, Sanjay R.; Davitz, Michael A.; Rifkin, Daniel B.; Wilson, E. Lynette

CS Med. Cent., New York Univ., New York, NY, 10016, USA

SO Blood (1994), 83(8), 2115-25
CODEN: BLOOAW; ISSN: 0006-4971

DT Journal

LA English

AB Basic **fibroblast growth factor** (bFGF) is a hematopoietic cytokine that stimulates stromal and stem cell growth. It binds to a **glycosylphosphatidylinositol** (GPI)-anchored heparan sulfate proteoglycan on human bone marrow (BM) stromal cells. The bFGF-proteoglycan complex is biol. active and is released by addn. of exogenous phosphatidylinositol-specific phospholipase C. In this study, the authors show the presence of an endogenous GPI-specific phospholipase D (GPI-PLD) that releases the bFGF-binding heparan sulfate proteoglycan and the **variant** surface glycoprotein (a model GPI-anchored protein) from BM cultures. An involvement of proteases in this process is unlikely, because released proteoglycan contained the GPI anchor component, ethanolamine, and protease inhibitors did not diminish the release. The mechanism of release is likely to involve a GPI-PLD and not a GPI-specific phospholipase C, because the release of **variant** surface glycoprotein did not reveal an epitope called the cross-reacting determinant that is exposed by phospholipase C-catalyzed GPI anchor cleavage. In addn., phosphatidic acid (which is specifically a product of GPI-PLD-catalyzed anchor cleavage) was generated during the spontaneous release of the GPI-anchored **variant** surface glycoprotein. The authors also detected GPI-PLD-specific enzyme activity and mRNA in BM cells. Therefore, the authors conclude that an endogenous GPI-PLD releases bFGF-heparan sulfate proteoglycan complexes from human BM cultures. This mechanism of GPI anchor cleavage could be relevant for mobilizing biol. active bFGF in BM. An endogenous GPI-PLD could also release other GPI-anchored proteins important for hematopoiesis and other physiol. processes.

NR

L4 ANSWER 9 OF 9 CA COPYRIGHT 2001 ACS

AN 117:83581 CA

TI Expression and immunochemical analysis of rat and human recombinant **fibroblast growth factor** receptor (flg) isoforms

AU Xu, Jianming; Nakahara, Mitsura; Crabb, John W.; Shi, Ergang; Matuo, Yuhsi; Fraser, Malcolm; Kan, Mikio; Hou, Jinzhao; McKeehan, Wallace L.

CS W. Alton Jones Cell Sci. Cent., Inc., Lake Placid, NY, 12946, USA

SO J. Biol. Chem. (1992), 267(25), 17792-803
CODEN: JBCHA3; ISSN: 0021-9258

DT Journal

LA English

NR

AB Potentially 96 splice **variants** among four genes that code for the human heparin-binding **fibroblast growth factor** receptor family complicate study of structure, metab., and function of single isoforms in mammalian cells. As an alternative, the authors expressed structural subdomains and isoforms of the flg receptor gene in bacteria and baculoviral-infected insect cells. The authors developed and characterized a panel of 16 isoform and domain-specific polyclonal and monoclonal antibodies. The panel of antibodies was used to distinguish mature **glycosylated** ligand-binding and kinase-active and -inactive recombinant isoforms in baculoviral insect cells and transfected mammalian cells and natural isoforms in rat prostate and humans liver cells. The results revealed a cell type-specific expression

of the flg gene and isoforms that result from combinations of splice variations. Reactive epitopes of monoclonal antibodies against both the three (.alpha.) and two (.beta.) Ig-like disulfide loop extracellular domain isoforms were mapped by cross-reactivity with synthetic polypeptide sequences and deletion mutants expressed in bacteria. The native .alpha. and .beta. receptor isoforms differed in display of shared epitopes and suggested that the NH2-terminal Loop I and COOH-terminal Loops II and III of the .alpha. isoform are interactive. Although the common Loops II and III appear qual. sufficient for ligand binding, the results suggest that tertiary relationships among loops in the three and two loop isoforms are distinct and, therefore, the two isoforms may have distinct activities. Spatial models for arrangement of Ig-like loops in the extracellular domain of the two isoforms are presented.

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